Acute carpal tunnel syndrome is an uncommon diagnosis most often related to blunt trauma requiring immediate surgical decompression to avoid serious sequelae. Patients who present with bleeding-related acute carpal tunnel syndrome tend to have severe pain, rapid onset of swelling, and neurologic symptoms that appear early and progress rapidly secondary to mass effect. Acute carpal tunnel syndrome can occur in anticoagulated patients spontaneously or after minor trauma.

This article describes a case of a 57-year-old man with progressive pain and paresthesias in the median nerve distribution after reaching for a picture frame. He was taking dabigatran, a direct thrombin inhibitor, for atrial fibrillation. He developed acute carpal tunnel syndrome secondary to spontaneous bleeding into the carpal canal and flexor tenosynovium with hematoma formation requiring surgical decompression. He reported immediate pain relief postoperatively, had no further bleeding complications, and regained full median nerve function within 2 months.

Dabigatran has gained recent popularity for the treatment of atrial fibrillation. Unlike warfarin, its use does not involve regular laboratory monitoring or dose titration. The risks and benefits of dabigatran should be considered carefully by the prescriber, particularly in patients taking medications that may alter its metabolism. Aspirin and nonsteroidal anti-inflammatory drugs may have effects similar to dabigatran and may increase the risk of bleeding problems. Should acute carpal tunnel syndrome occur, the authors recommend prompt surgical decompression rather than conservative management. The modification of anticoagulant therapy should be considered on a case-by-case basis.

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Acute carpal tunnel syndrome is an uncommon diagnosis most often related to blunt trauma. Unlike chronic carpal tunnel syndrome, acute carpal tunnel syndrome requires immediate surgical decompression to avoid serious sequelae. Other potential etiologies of acute carpal tunnel syndrome include hemorrhagic, vascular, and bleeding disorders, along with chronic and acute rheumatologic conditions.

Acute carpal tunnel syndrome is characterized by unrelenting pain and dysesthesia in the median nerve distribution secondary to a rapid increase in pressure in the carpal tunnel. It can be differentiated from chronic carpal tunnel syndrome by the rapid onset of severe symptoms and by its progressive course over hours rather than weeks or months. The carpal tunnel behaves like a closed compartment and is intolerant of space-occupying lesions; symptoms quickly develop as a result of increased pressure in the canal.

Patients who present with acute carpal tunnel syndrome associated with bleeding tend to have more severe pain, rapid onset of swelling, and neurologic symptoms that appear early and progress rapidly secondary to mass effect. If acute carpal tunnel syndrome is on the differential diagnosis, Mack et al reported that patients may be treated with nonsurgical measures, such as elevation, cast or dressing release, and observation for 2 hours. If these measures fail to relieve symptoms, surgeons should consider measuring carpal tunnel pressures directly with a wick catheter or other device to distinguish acute carpal tunnel syndrome from a median nerve contusion. When pressures exceed 40 mm Hg, carpal tunnel release should be performed within 8 hours of the onset of symptoms. If the diagnosis is obvious, proceeding to immediate surgical decompression without measuring compartment pressures is appropriate.

**Case Report**

A 57-year-old right-hand-dominant man presented to the emergency room with unrelenting left hand pain over the past 24 hours. He reported reaching for a picture frame with his left hand, feeling 1 of his fingers lock up, and developing pain and swelling of the hand over the next several hours. He developed progressive numbness and tingling in the median nerve distribution over the next 8 hours. His pain then progressed into the radial aspect of his hand, thumb through ring fingers, and volar proximal forearm. The pain was not relieved by position, rest, elevation, or analgesics.

His medical history was significant for hypertension and paroxysmal nonvalvular atrial fibrillation diagnosed 2 months previously and treated with cardioversion. He was placed on 150 mg of dabigatran twice daily and 81 mg of aspirin once daily. Other medications included verapamil and flecainide. He reported no symptoms of atrial fibrillation following cardioversion.

Clinically, he was afebrile, and his vital signs were stable. All of his laboratory results were within normal limits, and his international normalized ratio was 1.1. Examination revealed marked swelling and ecchymosis of the volar aspect of his left wrist that tracked into the mid-palm. He had no sensory perception to a 5.61 Semmes-Weinstein monofilament; however, he perceived deep pressure in the median nerve distribution. Radial and ulnar nerve function was grossly intact. He had mild pain with passive and active range of motion of his fingers. His radial pulse was palpable, and his capillary refill was brisk. Radiographs of the wrist were negative for osseous abnormalities but showed volar soft tissue swelling (Figure).

A diagnosis of atraumatic acute carpal tunnel syndrome was made, and the patient underwent emergent open carpal tunnel release under general anesthesia and under tourniquet control. When the transverse ligament was incised, a significant amount of liquefied hematoma existed. The release was completed, and further hematoma was milked proximally and distally. A minimal amount of clotted blood was also expressed. Liquefied blood was also present in the flexor tenosynovium. The median nerve grossly appeared normal. No other masses existed in the canal. A distal forearm fasciotomy was performed, and the palmar fascia distal to the incision was released. No significant active bleeding occurred after tourniquet deflation. The skin was closed over a drain, and a dry dressing and volar splint were applied. The hand was elevated using a sling suspended from an intravenous pole overnight.

On postoperative day 1, the patient’s sensation was markedly improved, and his pain was significantly decreased. The
Drain was removed after minimal drainage. His swelling had decreased significantly, and normal skin creases had returned. His cardiologist stopped dabigatran, with aspirin restarted on postoperative day 5. The patient’s median nerve examination continued to improve. From an atrial fibrillation standpoint, he had undergone a recent 14-day CardioNet (Conshohocken, Pennsylvania) study 1 month before he developed acute carpal tunnel syndrome, during which he showed no evidence of atrial fibrillation. As of the most recent evaluation 3 months postoperatively, he remained off of dabigatran, with no further bouts of atrial fibrillation. Two months postoperatively, a full return of median nerve sensory and motor function occurred.

**Discussion**

The current patient’s hospital course generally followed Mack et al’s algorithm.4 To the current authors’ knowledge, no reports have been published in the literature of acute carpal tunnel syndrome in association with dabigatran use. The patient had no other risk factors, and the diagnosis was clear based on the physical findings of severe pain, swelling, ecchymosis, and the loss of median nerve function, necessitating urgent surgical release. The patient was considered to have sustained relatively minor vascular trauma in the synovial flexor sheath. The hematoma expanded quickly, compressing the median nerve and resulting primarily in a neuropraxic injury. Measuring his carpal tunnel compartment pressures was not necessary because the diagnosis was clear, and pressure measurements were not performed.

Several treatment approaches for acute carpal tunnel syndrome in anticoagulated patients have been reported.5-9 Black et al9 suggested not stopping therapy but, rather, maintaining an appropriate international normalized ratio for patients on prophylactic medication, whereas others suggest reversing the warfarin and following the patient nonoperatively.6 Although a temptation exists to treat these patients conservatively, the severity of their symptoms may preclude this option, and, in addition, early surgical intervention has shown rapid and full restoration of median nerve function.5,7 Patients in whom surgical decompression is delayed may have a more prolonged or incomplete recovery, with the possible complication of early reflex sympathetic dystrophy.6,7

Dabigatran is an oral direct thrombin inhibitor that has gained recent popularity. Its half-life is 12 to 17 hours, and unlike warfarin, it claims the benefit of not requiring regular laboratory monitoring.10 Several large, premarket clinical trials of dabigatran have been performed. A manufacturer-sponsored study from the RE-LY group evaluated the efficacy and safety of 2 different doses of dabigatran compared with warfarin in more than 18,000 patients with atrial fibrillation. Taking 150 mg of dabigatran twice daily was significantly more effective in preventing stroke or systemic embolization compared with taking warfarin. The 150 mg dose of dabigatran was associated with a similar risk of major hemorrhage compared with warfarin. Despite its efficacy, a larger percentage of participants on dabigatran discontinued their enrollment in the trial because of gastrointestinal symptoms.11 Another recent study showed that patients younger than 75 years with atrial fibrillation had lower risks of intracranial and extracranial bleeding with dabigatran compared with warfarin.12

Dabigatran has also been studied for the treatment of acute thromboembolism. A trial of more than 2500 patients by the RE-COVER study group demonstrated the noninferiority of dabigatran when compared with warfarin in the treatment of acute venous thromboembolism, with a similar rate of major bleeding and a lower rate of minor bleeding.13 However, more patients stopped taking the drug due to gastrointestinal side effects.13 Dabigatran has been evaluated for use as a prophylactic anticoagulant for patients undergoing orthopedic procedures. Two studies comparing dabigatran to enoxaparin have showed improved or equivalent efficacy in preventing thrombosis14,15; however, an increased bleeding risk existed in the higher-dose dabigatran group.

Although no laboratory monitoring is necessary with dabigatran, prescribing physicians should be aware of factors that can affect its efficacy. Fatty foods can delay the absorption of dabigatran.16 Stangier17 reported that the drug’s absorption may be moderately decreased if it is taken with a proton pump inhibitor. Drug excretion through p-glycoprotein pumps is slowed in patients taking common strong p-glycoprotein pump inhibitors, such as quinidine, rifampin, fluconazole, St John’s wort, verapamil, and amiodarone, thus raising the plasma levels of dabigatran.18 The current patient was taking verapamil, which could have raised the serum concentration of dabigatran. The patient was also taking aspirin; however, according to the RE-LY trial, no significant interactions or increased side effects were demonstrated in patients taking aspirin and dabigatran.11 Before including aspirin and other nonsteroidal anti-inflammatory drugs in the postoperative period, one should take into account the patient’s cardiovascular risks and talk with their cardiologist about alternative options for their anti-coagulation.

The current authors recommend withholding medications that can increase bleeding in the acute postoperative period.

**Conclusion**

The risks and benefits of dabigatran should be considered carefully by the prescribing physician, particularly in patients also taking aspirin or p-glycoprotein pump inhibitors. Patients should be informed of symptoms of excessive bleeding in the carpal tunnel, even with minor...
trauma. Should acute carpal tunnel syndrome occur, the authors recommend prompt surgical decompression rather than conservative management. Due to the transient effect of dabigatran and its short half-life, no need for further medical monitoring should exist other than a stay overnight. Modification of anticoagulant therapy postoperatively should be considered on a case-by-case basis, with alternatives discussed with their cardiologist.

**References**